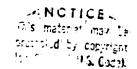


The Effect of Involuntary Smoking on the Occurence of **Chronic Obstructive Pulmonary Disease**

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Mainstream smoke (MS) is the tobacco smoke that is drawn through the butt end into the smoker's respiratory system. Sidestream smoke (SS) comes directly from the burning end of the cigarette. «Passive smoking», «involuntary smoking» and exposure to «environmental tobacco smoke» (ETS) are used synonymously in the literature to describe the exposure of non-smokers to elements of tobacco smoke generated by smokers [1, 2]. Despite qualitative differences between mainstream smoke and sidestream smoke, it has been customary to assume that exposures to ETS approximate a low-dose exposure to tobacco smoke. Since cigarette smoking is the major cause of Chronic Obstructive Pulmonary Disease (2022) mortifity and mortality in most countries of the world, and children of smoking parents have; (i) increased prevalence of reported respiratory symptoms (in sincreased fre quency of bronchitis and pneumonia early in the (iii) measurable though small differences in (iii) pulmonary function, when compared with children of non-smoking parents [3], an effect of passive smoking in adults should not be excluded. Having undertaken in the late 70's a study to explore the association between involuntary smoking and lung cancer [4], it was decided in 1982 to undertake a similar study to investigate the association between involuntary smoking and COPD in adults in Greece. The study was restricted to non-smoking ever-married women residents of the Greater Athens area. Among relatively older Greeks the prevalence of smoking is very high in men but relatively rare in women, making a passive smoking study in women, rather than in men, considerably more efficient. As in most of the studies concerning involuntary smoking and lung cancer, the present study evaluated exposure to passive smoking on the basis of the smoking habits of the husband. The underlying assumptions in this approach are (i) that a smoking husband is the main source of passive smoking for a relatively older woman; (ii) that information concerning the smoking behaviour of a husband is much more reliable than information concerning other sources of passive smoking; (iii) that non-smokers married to smokers are likely to be more tolerant towards other sources of passive smoking; and (iv) that smokers tend

This investigation was part of the Professorial Thesis of one of us (ST) and University rules did not allow the publication of the results before the thesis was submitted to the Medical School at the fall of 1987.

Material and Method

Between January 1982 and December 1983, 137 evermarried women 40-79 years old were admitted to the Seventh Clinic of the Teaching Hospital for Respiratory Diseases in Athens with a diagnosis of COPD. Single women were not included in the study since their life-styles in Greece are sufficiently different from those of married women. Women younger that 40 and older than 80 were not included in the study because very young women could not have been exposed to their husband's smoking for a period long enough to acommodate the postulated latency of COPD, whereas information coming from very old women could be less reliable than information provided from younger women. Women were included in the study if they were hospitalized for the first time with principal diagnosis of COPD. The case series included patients who were presented with dysphoeas on exertion and expectoration for at least 3 years, and who were found in the laboratory to have obstructive or mixed type reduction of pulmonary function by at least 20% in forced expiratory volume in 1s, without improvement after bronchodilatation [3]. Seven women with a history of bronchial asthma or familiar history of pulmonary emphysema were excluded from the study whereas two additional women refused to be inerviewed. Among the remaining 128 women of the index series 18 were current and 7 have been past smokers and were excluded from further analysis. N (Smoking status was ascertained with reference point) one year before the interview; thus those who stopped to smoking six months before the interview were counted as current smokers). The remaining 103 women have twice denied that they have ever been smokers, once during their routine history-taking and then categorically at interview in the study. Controls were 206 evermarried women of similar age who were visitors (friends or relatives) of patients at the hospital during the same period. Six women refused to be interviewed and among the remaining 200, 7 were current and 14

to cluster.

Following earlier Greek experience as well as international practice [4-7] the smoking habits of the husband were taken as indicators of exposure to environmental tobacco smoke. More specifically, exposure variables were based on husband's smoking habits in terms of amount (non-smoker, ex-smoker of any specified amount, and current smoker by average daily number of cigarettes; smoking of other forms of tobacco is rare in Greece), duration (beginning from the date of marriage or start of smoking by the husband, whichever came later, and ending at the date that the husband died or stopped smoking or current date, whichever came first), or their product (total number of cigarettes smoked).

In order to control potential confounding effects data were also collected from cases and controls with respect to schooling years of the woman and her husband, place of residence (urban or rural) before permanent setlement in Athens (all women were permanent residents of Athens) and occupation (housewife, other). Analysis was done with standard stratification procedures [8, 9].

Results

Table 1 shows univariate characteristics of cases and controls with respect to age in years, woman's schooling in years, husband's schooling in years, previous

residence (rural, i e less than 10000, or urban) and woman's occupation (housewife or other). There are clearly none significant differences with respect to woman's schooling, husband's schooling and previous residence, but age and woman's occupation are potential confounders and should be controlled in the analysis.

Tab. 1. Univariate characteristics of 103 non-smoking ever-married women, first hospitalized with chronic obstructive pulmonary disease, and 179 non-smoking ever-married visitor controls, with respect to potential confounding factors

Risk factors		COPD		Controls		Difference or heterogeneity	
Age (years):		N	%	N	%	(P two tails)	
40-49		21	20:4	44	24.6		
50-59		22	21.4	67	37.4		
60-69		26	25.2	50	27.9	0.001	
70-79		34:	33.0	18	10.1		
Woman's sch	iooling						
-5	≤ 5 years	56:	54.4	92	51.4		
6+	> 5 years	47	45.6:	87.	48.6	0.6	
Husband's si	chooling						
-5	€ 5 years:	34	33.0	52	29.1		
6+	> 5 years	69	67.0	127	70.9	0.5	
Previous res	idence						
Rural		41	39.8	62	34.6		
Urban		62	60/2:	117	65.4	0:4	
Woman's oc	cupation						
Housewife.	•	55	53.4	123	68.7		
Other		48:	46.6	56	311.3	0.01	

Tab. 2. Distribution of 103 non-smoking ever-married women, first hospitalized with chronic obstructive pulmonary disease, and 179 non-smoking ever-married visitor controls, by age, occupation and average daily number of cigarettes smoked by the husband

Age	Occupation		Everage daily number moked by the husband				-
			Non-smoking	ex-smoker	1-20	21+	Total
1019	Housewife	Cases	0	1	8	5	1-4
		Controls	9.	5	3!	13	30
40-49 Working	Working	Cases	li	1:	01	5.	7
	•	Controls	2	3	3	6:	1-
50-59	Housewife	Cases	3.	1.	5	0:	9
		Controls	7	14	13	16	50
50-59	Working	Cases:	1.	1	4	7.	13
	•	Controls	7.	2:	3	5	17
60-69 1	Housewife	Cases:	2:	1	5	5:	13
		Controls	3	13	6	9	31
60-69 Wo	Working	Cases	2:	2:	4	5:	13
	- -	Controls	2:	10	2	5:	19
70-79 Hou	Housewife	Cases	3.	7.	7.	2	19
		Controls	1	3	3	5	12
70-79	Working	Cases	1	4	2	8	15
	•	Controls	2	2	1.	1.	6
Total		Cases	13.	18	35	37	103
		Controls	33	52	34:	601	179
Relative ri	sk (crude)		1/0	0.9	2.6	1.6:	
Relative risk (Mantel-Haenszel)		1:0	0.6	2.4	1.4		
(90% Confidence Limits)			0.3-1.3	1.3-4.5	0.8-2.6		
	nd. crude = 3.57	Pone tail =	0.03				
	nd. Mantel extensio						

Tab. 3. Distribution of 103 non-smoking ever-married women, first hospitalized with chronic obstructive pulmonary disease, and 179 non-smoking ever-married visitor controls, by age, occupation and total life-long number of cigarettes smoked by the husband (in thousands).

Age:	Occu <u>p</u> ation	Cases or: Controls	non-smoker	<:300	Life-long total n cigarettes smoke > 300	number of ed by the husband (thousands) Total
40-49.	Housewife	Cases	0:	13	1	14
		Controls	9:	14	7	30:
4049.	Working	Cases	1	5	1.	7
	-	Controls	2:	11	1.	14.
50-59	Housewife	Cáses	3	5	1.	9.
		Controls	7	32	11.	50
50-59	Working	Cases	1	7.	5	13
		Controls	7	6	4:	17
60-69	Housewife	Cases	2	3.	8	13
		Controls	3	19.	9:	31
60-69	Working	Cases	2	5:	6:	13
		Controls	2	111	6:	19
70-79	Housewife	Cases	3	9	7	19
		Controls	1	10	1	12
70-79	Working	Cases	1	5:	9:	15
		Controls	2	1:	3	6
Total		Cases:	13.	52	38	103
		Controls	33	104	42	179
Relative n	sk (crude)		1.0	1.3	2.3	
Relative risk (adjusted):		1.0	1.3	1.8		
(90% Confidence Limits)			0:8-2:4!	0.9-3.6		

 x^2 for trend1 crude = 5.58 Pone tail = 0.01. x^2 for trend1 Mantellextension = 3.44 Pone tail = 0.03

Tables 2 and 3 show the distributions of cases and controls by age, occupation and average daily number of cigarettes smoked by the husband (table 2), or total life-long number of cigarettes smoked by the husband in thousands (table 3). In both instances there are statistically significant (one tail P<0.05) linear trends between amount of tobacco smoked by the husband and risk of (hospitalization for): COPD. Thus, it appears: that exposure to environmental tobacco smoke may contribute to the development of COPD with an associated relative risk of about 2.

Discussion

Many epidemiologic investigations have firmly established that exposure of children a sufficient parental lobacco smoke, usually attributable to the stocking habits of their parents, has clear directs on their respiratory system. The relevant studies more than 25 by now have recently being reviewed [6, 7, 10, 11]. By contrast, studies in adults have been fewer and their results less clear-cut. Most of these studies have focused on pulmonary function rather than on clinical syndromes, and among them about half have found significant evidence that exposure to environmental tobacco smoke has adverse pulmonary effects [12–16], whereas in the other half such evidence was not apparent [17–21].

Very few studies have examined the association, if any, between exposure to environmental tobacco smoke and development of COPD as a clinical entity—for instance no such study has been included in the

major review volumes published by the National Research Council [7] and the Surgeon General [6] or the recent review paper by Fielding and Phenow [11]. Yet investigations of a possible link between exposure to ETS and chronic obstructive pulmonary disease should be particularly promising for at least three reasons: chronic obstructive pulmonary disease is one of the entities most strongly related to active smoking [3]; a clinical COPD is by definition an extreme expression of the pathophysiological changes manifested as disturbances of pulmonary function(s) and it is a general epidemiologic principal that extreme points designs are inherently more powerful [22]; since individuals more susceptible to the imitating effects of cigarette smoke on the lower respiratory tract are more likely to be non-smokers [7], a population of non-smokers passively exposed to ETS would be more likely to include susceptible individuals who in turn would be more likely to develope the constellation of symptoms and signs of COPD.

There are in the literature short reports of, or references to, three studies concerning the association between passive smoking and COPD. In the cohort study of Hirayama in Japan, a statistically significant relative risk of 1.6 was found in non-smoking women passively exposed to the tobacco smoke of their husband [23]. In papers presented in 1987 in the meetings of the Society for Epidemiologic Research and the International Epidemiology Association, Sandler and her colleagues reported that in their cohort an increased relative mortality from respiratory diseases

was found among non-smokers passively exposed to tobacco smoke, compared to non-smokers not so exposed [24]. There has been also a brief publication of preliminary results of the present study [5] (there are slight differences between the figures reported earlier and those shown here, due to recoding and use of different strata in the standardization procedures). The results of the present study taken together with those of the two cohort studies [23, 24] and the collective findings of the epidemiologic studies exploring the association between exposure to ETS and various parameters of pulmonary function, suggest that passive smoking may contribute to the development of COPD. The reported association is unlikely to be explained in terms of misclassification of current or past smokers as non-smolecus [25]. Furthermore, relatives risks figures generated from empirical studies should be adjusted upwards, since few; if any, subjects are actually completely offexposed to passive smoking [26]. Lastly, random missiassification of ETS exposure and misspecification of biologic latency are likely to generate systematic underestimation of the true underlying relative risk.

Summary

One:hundred and three ever-married women with newly diagnosed Chronic Obstructive Pulmonary Disease (COPD), who have never smoked, and 179 ever-married women who were visiting friends or relatives at the same hospital during the same period and have never smoked, were interviewed regarding the smoking habits of their husbands. There was statistically marginally significant difference between the COPD cases and the controls with respect to their husband's smoking habits. The association was irregular with respect to daily number of oigarettes smoked but there was a smooth dose response curve with respect to life long total number of cigarettes smoked, with women whose husband smoked more that 300 thousand cigarettes having a relative risk of 1.8 (90% confidence interval of 0.9-3.6) compared to women whose husband has never smoked. These findings, and converging related evidence, indicate that exposure to environmental tobacco smoke may be a risk factor for the development of COPD:

Résumé

Effets de la famée passive sur la survenue d'un syndrome bronchoobstructif chronique:

Cent-trois femmes, manées de longue date, n'ayant jamais fumé, et chez: qui un syndrome broncho-obstructifi chronique (SOC) a été récemment diagnostiqué, ont été interrogées pour ce qui concerne les habitudes tabagiques de leur maris: Leurs réponses furent comparées à celles de 179 femmes, également manées de longue date et non-fumeuses, qui s'étaient rendues dans le même hôpital pour rendre visite à un parent ou ami. Les habitudes tabagiques des maris sont différentes entre les cas de SOC et les contrôles, mais les différences ne sont que marginalement statistiquement significatives. L'association est inconstante par rapport au nombre quotidien: de cigarettes fumées, cependant la courbe dose-réponse en fonction du nombre total de cigarettes fumées au cours de la vie montre une progression régulière: Les femmes dont les maris ont fumé plus de 300000 cigarettes ont un risque relatif de développer un SOC de 1.8 untervalle de confiance 90%: 0.9-3.6) par rapport aux femmes dont les maris n'ont jamais fumé. L'association décrite ici, ainsi que d'autres preuves concordantes, indiquent que l'exposition passive à la tumée de cigarettes peut être un facteur de risque pour la survenue du SOC.

Zusammenfassung

Passivrauchen und chronisch obstruktive Lungenkrankheiten

In einer Fall-Kontroll-Studie wurden 103 Patientinnen mitichronisch. obstruktiver Lungenkrankheit erfasst, die nie geraucht hatten und zur Zeit der Studie verheiratet waren oder es zumindest frühereinmal gewesen waren. Als Kontrollen dienten 179 Frauen auf Krankenvisite im selben Spital, die ebenfalls nie geraucht hatten und auch zumindest einmal verheiratet gewesen waren. Verglichen wurden die Rauchgewohnheiten der Ehemanner. Der einfache Vergleich Raucher-Nichtraucher ergab eine schwach signifikante Differenz, ein eindeutiger Zusammenhang mit der taglich gerauchten Anzahl Zigaretten fand sich jedoch nicht. Eine dosisabhangige Beziehung zeigte sich bei der im Lieben insgesamt gerauchten Anzahl Zigaretten: das relative Risiko war für Frauen, deren Ehemänner mehr als 300000 Zigaretten geraucht hatten 1.8 im Vergleich zu Partnerinnen von Nichtrauchern (90% Vertrauensinterval) 0.9-3:6). Diese Resultate konnten ein: Hinweis dafur sein, dass Nichtrauchen ein Risikofaktor für chronisch obstruktive Lungenkrankheiten sein konnte:

References

- [1] Săracci R. Riboli E. Passive smoking and lung cancer: current evidence and ongoing studies at the International Agency for Research on Cancer: Mutat Res 1989; 222: 117-27.
- [2] Trichopoulos D. Passive smoking and lung cancer. Scandil Soc Med 1988; 16: 75-79.
- [3] Office on Smoking and ealth. The health consequences of smoking: Chronic obstructive lung disease. A Report of the Surgeon General. Rockville: Md.: Office on Smoking and Health. 1984. (DHHS Publication No. (PHS) 84-50205).
- [4] Trichopoulos D. Kalandidi A. Sparos L. MacMahon B. Lung cancer and passive smoking. Int J Cancer 1981; 27: 1-4.
- [5] Kalandidi A. Trichopoulos D. Harzakis A. Tzannes S. Saracci R. Passive smoking and chronic obstructive lung disease. Letter to the Editor: Lancet 1987: ii: 1325-26.
- [6] Office on Smoking and ealth. The health consequences of involuntary smoking. A Report of the Surgeon General. Rockville. Md.: 1986 (DHH5 Publication No. CDC 87-8398)
- [7] National Research Council. Committee on passive smoking. Board on Environmental Studies and Toxicology. Environmental tobacco smoke: Measuring exposures and assessing health effects. Washington. DC:: National Academy Press, 1986.
- [8] Mantel N. Haenszel W. Statistical aspects of the analysis of data from retrospective studies of disease. J Natl Cancer Inst. 1959: 22:719-48.
- [9] Mantel N. Chi-square tests with one degree of freedom: extention: of: the: Mantel Haenszel procedure: JASA, 1963, 59-690-700.
- [10] Froggan P. chairm. Fourth report of independent Scientific Committee on Smoking and Health. London: HMSO. 1988.
- [11] Fielding J. Phenow K.: Health effects of involuntary smoking. N Engl J Med 1988; 319: 1452-60.
- [12] White JR. Froeb HF. Small-airways disfunction in nonsmokers chronically exposed to tohacco smoke. N Engl J Med 1980; 302: 720-23.
- [13] Kauffmann F. Tessier JF. Oriol P. Adult passive smoking in the home environment: A risk factor for chronic airflow limitation. Am J Epidemiol 1983; 117: 269–80.
- [14] Kauffmann F. Dockery DW. Speizer FE. Ferris BG. Respirations symptoms and lung function in women with passive and active smoking. Am Rev Respir Dis 1986; 133. A157.
- [15] Brunekreef, B. Fischer P. Remijn B, Van der Lende R. Schouten J. Quanjer P. Indoor air pollution and its effection-pulmonary function of adult nonsmoking women: 3. Passive smoking and pulmonary function. Int J Epidemiol 1985; 14: 227-30.
- [16] Svendsen KH. Kuller LH. Neaton JD: Effects of passive smoking in the Multiple Risk Factor Intervention Trial. Circulation 1985; II: No.4
- [17] Schilling RSF, Letai AD. Hui SE. Beck GJ. Schoenberg J.B. Bouhuys AH: Liung function, respiratory disease and smoking in families. Am J Epidemiol: 1977; 106: 274-83.

- [18] Comstock GW. Meyer MB. Helsing KJ, Tockman MS. Respiratory effects of household exposures to tobacco smoke and gas cooking. Am Rev Respir Dis 1981: 124: 143-48.
- [19] Schenker MB. Samei JM. Speizer FE. Effect of cigarette tar content and smoking habits on respiratory symptoms in women. Am Rev Respir Drs 1982: 125: 684-90.
- [20] Jones JR: Higgins ITT. Higgins MW. Keller JB: Effects of cooking fuels on lung function in nonsmoking women: Arch Environ Health/1983; 38: 219-22.
- [21] Kenmer M. Triebig G. Weltle D. The influence of passive smoking on pulmonary function: A study of 1351 office workers. Prev. Med 1984; 13: 656-69.
- [22] Miettnen O: Theoretical epidemiology: Principles of occurence research immedicine. New York: Wiley Medical, 1985.
- [23] Hirayma T. Lung cancer in Japan: Effects of nutrition and passive smoking. In: Mizell Mi Correa P. eds. Lung cancer: Causes and prevention. Weinheim: Verlag Chemie International. 1984: 183.
- [24] Sandler DR. Helsing KJ. Shore DL. Comstock GW. All cause mortality among nonsmokers with household smoke exposure. Abstracts of presentations at meetings of the Society for Epidemiologic Research and International Epidemiology Association.

- [25] Wald N.: Nanchahal K. Thompson S. Cuckle H. Does breathing other: people's tobacco-smoke cause lung cancer? Br. Med J 1986; 293: 1217-22.
- [26] Vuduc C. Quantitative aspects of passive smoking and lung cancer. Prev Med 1984; 130: 187-92.

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